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The role of the ventrolateral caudoputamen in predatory hunting

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ABSTRACT

The ventrolateral caudoputamen (VLCP) is well known to participate in the control of orofacial movements and forepaw usage accompanying feeding behavior. Previous studies from our laboratory have shown that insect hunting is associated with a distinct Fos up-regulation in the VLCP at intermediate rostro-caudal levels. Moreover, using the reversible blockade with lidocaine, we have previously suggested that the VLCP implements the stereotyped actions seen during prey capture and handling, and may influence the motivational drive to start attacking the roaches, as well. However, considering that (1) lidocaine suppresses action potentials not only in neurons, but also in fibers-of-passage, rendering the observed behavioral effect not specific to the ventrolateral caudoputamen; (2) the short lidocaine-induced inactivation period had left a relatively narrow window to observe the behavioral changes; and (3) that the restriction stress to inject the drug could have also disturbed hunting behavior, in the present study, we have examined the role of the VLCP in predatory hunting by placing bilateral NMDA lesions three weeks previous to the behavior testing. We were able to confirm that the VLCP serves to implement the stereotyped sequence of actions seen during prey capture and handling, but the study did not confirm its role in influencing the motivational drive to hunt. Together with other studies from our group, the present work serves as an important piece of information that helps to reveal the neural systems underlying predatory hunting.

1. Introduction

Predatory hunting is an innate behavioral response critical for the survival of animals [1]. To study predatory behavior, we have used insect hunting, which appears to be an ideal condition to investigate predatory behavior in rats [2]. In this paradigm, roaches have been chosen as suitable prey, since they are relatively innocuous and easily overcome. Indeed, rats display an innate pattern of prey hunting similar to the one seen in small insectivores. This stereotyped sequence of actions is even present in rats hunting for the first time, supporting the idea of an innate motor program to capture and handle the prey, which certainly increases hunting efficiency. Previous studies from our laboratory have shown that insect hunting is associated with a distinct Fos up-regulation in the ventrolateral caudoputamen at intermediate rostro-caudal levels [2]. The ventrolateral caudoputamen is largely known to be involved in controlling orofacial movements and forepaw usage accompanying feeding behavior [3,4]. Moreover, the view that the striatum is a key structure for choosing actions or sequence of actions is prevalent in most of the basal ganglia literature [5–7]. Thus, the striatum has been pointed at as a key structure involved in sequential behavior, like learned serial reaction time [5], grooming and other kinds of stereotyped behaviors [8].

In a previous study, we have shown that the lidocaine-induced reversible inactivation of the rat ventrolateral caudoputamen causes severe deficits during prey hunting [9]. During bilateral blockade of the ventrolateral caudoputamen, the animals showed a significantly longer latency to start capturing the prey and an awkward motor pattern during prey capture, using mostly the mouth, with little forepaw assistance, resulting in deficient prey capture. However, considering that lidocaine suppresses action potentials not only in neurons of the target structure, but also in the fibers-of-passage, this effect may be not specific to the ventrolateral caudoputamen [10]. Another drawback of this study was the short lidocaine-induced inactivation period, which left a relatively narrow window to observe the behavioral changes. Moreover, the restriction stress to inject the drug may also have disturbed hunting behavior.

In the present study, to circumvent these problems, we have examined how bilateral cytotoxic lesions placed in the ventrolateral caudoputamen three weeks previous to the testing procedures would influence prey hunting. Overall, the present findings support the idea of the ventrolateral caudoputamen as a critical site to organize the stereotyped sequence of actions during prey hunting.

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2. Materials and methods

2.1. Animals and housing

Adult male Wistar rats ($n=29$), weighing about 300 g and obtained from the Federal University of Parana State (UFPR) breeding facilities, were used in the present study. The animals were kept under controlled temperature (23 °C) and illumination (12 h cycle) in the animal quarters, and had free access to water and standard laboratory diet. Experiments were carried out in accordance with the National Institutes of Health Guide for the Care and Use of Laboratory Animals (NIH Publications No. 80-23, 1996). All experimental procedures had been previously approved by the Institutional Animal Care and Use Committee of the UFPR (protocol number 292). In the present study, we attempted to minimize the number of animals used and their suffering.

2.2. Experimental groups and surgical procedures

Twenty nine rats were divided into 4 experimental groups, namely, control non-operated (n = 8), ventrolateral caudoputamen (VLCP) sham- (n = 7) and NMDA-lesioned (n = 7), and dorsomedial caudoputamen (DMCP) NMDA-lesioned (n = 7) groups. Twenty minutes before surgery, the animals received atropine sulfate (0.4 mg/kg, i.p., Sigma-Aldrich, São Paulo, SP, Brazil) and penicillin G-procaine (20.000 Uin 0.1 ml, i.m., Bristol-Myers Squibb, São Paulo, SP, Brazil), and were anaesthetized with Equithesin (3 ml/kg, i.p.; 1% sodium thiopental, 4.25% chloral hydrate, 2.13% magnesium sulfate, 42.8% propyleneglycol and 3.7% ethanol in water). The VLCP and DMCP NMDA-lesioned groups received 0.8 μl of a 0.2 M solution of N-methyl-D-aspartate (NMDA, Sigma Chemical Co., St. Louis, MO, USA) bilateral injections into the ventrolateral caudoputamen (AP, + 0.5 mm from the bregma; ML, 3.7 mm from the midline; DV, − 0.7 mm from the skull) or the dorsomedial caudoputamen (AP, + 0.5 mm from the bregma; ML, 2.2 mm from the midline; DV, − 0.4 mm from the skull). The rats of the sham-lesioned group received 0.8 μl of saline (NaCl 0.9%) bilaterally into the ventrolateral caudoputamen (see coordinates above). Animals recovered for 3 weeks after surgery, prior to the predatory hunting test session. Animals from all experimental groups had their body weight, food and water intake measured (either one day before and six days after the surgery for the sham- and NMDA-lesioned animals, or during a seven-day period two weeks before the behavioral testing for the control non-operated group)

2.3. Experimental apparatus and behavioral testing

One week before the testing procedure, animals were individually housed into a Plexiglas cage (50 cm × 35 cm × 16 cm) and handled repeatedly by the same investigator who conducted the behavioral tests.

Animals were food deprived 24 h before the hunting sessions, which were carried out between 9:00 and 12:00 h, during the light phase of the cycle. In the hunting session, animals were induced to hunt by a simultaneous introduction, into the hunting cage, of five mature intact cockroaches (*Picnocellus surinamensis*), raised for this purpose in our laboratory. The hunting behavior was videotaped for further behavioral analysis.

2.4. Behavioral analysis

Behaviors were scored by a trained observer using the ethological analysis software "The Observer" (version 5.0; Noldus Information Technology, Wageningen, The Netherlands). For the behavioral analysis of predatory hunting, we first determined the latency to start hunting, and in the subsequent 15 min, we carefully examined the motor pattern to capture, hold and kill the prey, recording the following behavioral parameters: the number of attempts to capture the prey, number of successful captures (when the animals could hold the prey for more than 10 s), time spent eating, time spent displaying other behaviors than hunting (i.e., grooming, general exploratory activity and resting).

2.5. Open-field test

In order to assess possible effects of the lesions on motor activity and emotional behaviors, open-field test was performed 1 day after the hunting session. In the open-field test, rats were allowed to freely explore an open field for 5 min. The open-field apparatus consisted of a white 100 cm diameter circular arena with 50 cm high walls. The floor had three concentric circles and 12 black radius lines between the external and mid circles. The following items were recorded: total number of lines crossed (crossings), crossing in the 2 central circles, number of rearings, time spent in freezing (complete immobility, wide open eyes and muscle rigidity) and grooming, as well as the number of fecal boluses.

2.6. Histology

On completion of the open-field test, all rats were injected with sodium pentobarbital (Cristália; 40 mg/kg, i.p.) and perfused transcardially with a solution of 4.0% paraformaldehyde in 0.1 M phosphate buffer at pH 7.4; the brains were removed and left overnight in a solution of 20% sucrose in 0.1 M phosphate buffer at 4 °C. The brains were then frozen and four series of 30 μm sections were cut with a sliding microtome in the frontal plane. Sections were mounted on gelatin-coated slides and stained with thionin to serve as a reference series for cytoarchitectonic purposes. The sections were examined by using the 10× objective of a Nikon Eclipse 80i (Nikon Corporation, Chiyoda-Ku, Tokyo-To, Japan) microscope equipped with a Nikon digital camera DXM1200F (Nikon Corporation). The parcellation of the brain areas examined in the present investigation followed *The Brain Maps: structure of the rat brain* [11].

2.7. Statistical analysis

Homogeneity of variances was tested with the Levene's test. The open-field test behavioral data and the body weight, food and water intake measurements were analyzed by ANOVA followed by the Newman–Keuls test. The hunting behavioral data were analyzed using a non-parametric analysis of variance (Kruskal–Wallis test). For pairwise comparisons, we conducted Dunn's multiple comparison test in order to isolate the respective effect. Average results are expressed as mean ± SEM throughout the text.

3. Results

The parameters described above for NMDA injections resulted in lesions characterized by neuronal cell loss filled with gliosis (Fig. 1). In six animals, the lesions were centered bilaterally in the ventrolateral caudoputamen (VLCP), extending through the intermediate two thirds of its rostro-caudal axis (Fig. 2). The lesions tended to be largely circumscribed to the caudoputamen, and some lesions spread, to a small degree, to immediately adjacent sites, including the dorsal endopiriform nucleus, substantia innominata and magnocellular preoptic nucleus (Fig. 2). In one animal that received NMDA injection in the VLCP, the lesion was too extensive, including a large part of the piriform cortex, and this animal was excluded from the study. In 5 animals, the lesions were centered bilaterally in the dorsomedial caudoputamen (DMCP), extending through the intermediate two thirds of its rostro-caudal axis (Fig. 2). In other two animals, the NMDA

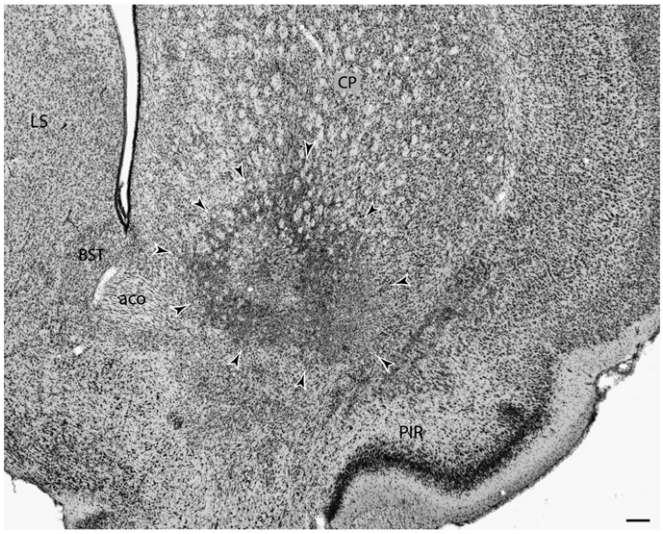

Fig. 1. Photomicrograph of transverse thionin-stained section illustrating the extent and appearance of a lesion centered in the ventrolateral caudoputamen (arrowheads). Abbreviations: aco—anterior commissure; BST—bed nuclei of the stria terminalis; CP—caudoputamen; LS—lateral septum; PIR—Piriform cortex. Scale bar: 200 μm.

injections aimed at the DMCP resulted in unilateral lesions, and those animals were excluded from the study.

As shown in Fig. 3, after surgery, the rats of the DMCP NMDA-lesioned and VLCP (sham- and NMDA-lesioned) groups presented a significant loss of body weight compared to the control (non-operated) rats [group effect $F(3,22)=6.07$, $P<0.01$; day effect $F(8,176)=28.44$, $P<0.001$; group × day interaction $F(24,176)=10.49$, $P<0.001$; $P<0.05$, post-hoc Newman–Keuls test], but no significant difference was observed comparing the NMDA-lesioned groups with the sham-operated rats (post-hoc Newman–Keuls test). In the same manner, surgery affected the food and water intake. In relation to control non-operated rats, during 2 days after the surgery, the sham-lesioned, DMCP NMDA- and VLCP NDMA-lesioned rats restricted significantly their food intake [group effect $F(3,22)=8.24$, $P<0.001$; day effect $F(6,132)=39.64$; interaction group × day $F(18,132)=6.24$, $P<0.001$, two-way ANOVA; $P<0.001$, Newman–Keuls test] and water intake [group effect $F(3,22)=5.71$, $P<0.01$, day effect $F(8,176)=54.06$, $P<0.001$; group × day interaction $F(24,176)=1.31$, $P=0.15$, two-way ANOVA; $P<0.001$, post-hoc Newman–Keuls test], but no significant difference was observed among the operated groups ($P>0.05$, post-hoc Newman–Keuls test). However, after surgery, these groups increased their food and water intake, and three days after surgery, no significant difference was observed in relation to the non-operated rats ($P>0.05$, post-hoc Newman–Keuls test).

A non-parametric ANOVA (Kruskal–Wallis test) revealed that, among the experimental groups, there was no difference in the latency to start hunting [$H(3,25)=0.34$; $P=0.34$] (Fig. 4A). Animals from all groups started chasing the prey shortly after they had been delivered into the testing box, orienting themselves very efficiently toward the moving prey, while trying to capture them. In sharp contrast, the Kruskal–Wallis test indicated significant differences among the experimental groups for the ratio of successful captures [$H(3,25)=14.33$; $P<0.01$], and pairwise comparisons (Dunn's multiple comparison test) revealed that VLCP NMDA-lesioned animals presented a significant drop in the ratio of successful captures when compared to the other groups ($P<0.05$), which did not differ statistically ($P>0.05$) (Fig. 4B). Thus, animals from the control non-operated, VLCP sham-lesioned and DMCP NMDA-lesioned groups performed the capture using the mouth, assisted by the forepaws. These animals caught the prey very efficiently, presenting a close to one ratio between the number of successful captures and the total number of catching attempts (Fig. 4B). As the prey had been captured, animals held them firmly with the forepaws and delivered the killing bite, ripping off the roaches' head. After killing the prey, animals either started eating them right away or carried on hunting other prey to consume them afterwards. The rats usually took the killed roaches to a corner of the cage and tried to conceal the captured prey from other potential predators (dodging behavior), while eating them voraciously. It is noteworthy that these animals did not have a previous hunting experience, but performed the insect hunting quite well, displaying a rather stereotyped sequence of motor actions for chasing, capturing and killing the prey.

In sharp contrast, VLCP NMDA-lesioned animals were very ineffective when trying to catch the roaches. First, they usually tried to seize them with the forepaws, but repeatedly let them escape. To hold the prey, they used mostly the mouth, with little assistance from the forepaws. Moreover, VLCP NMDA-lesioned animals looked very clumsy

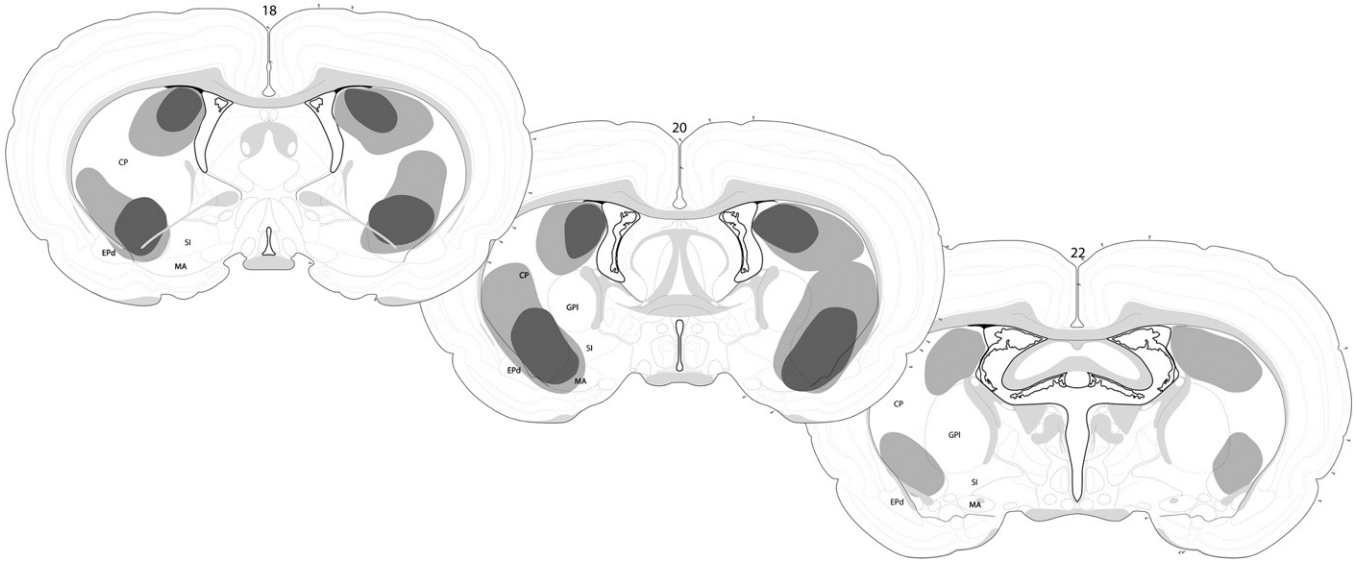

Fig. 2. Schematic representations along the rostro-caudal axis of the caudoputamen adapted from Swanson [1], showing the largest (dark gray areas) and smallest (black areas) NMDA lesions of the ventrolateral and dorsomedial caudoputamen. The midline numbers refer to the plate number of the Brain Maps [1]. Abbreviations: CP, caudoputamen; EPd, endopiriform nucleus, dorsal part; GPl, globus pallidus, lateral segment; MA, magnocellular preoptic nucleus; SI, substantia innominata.

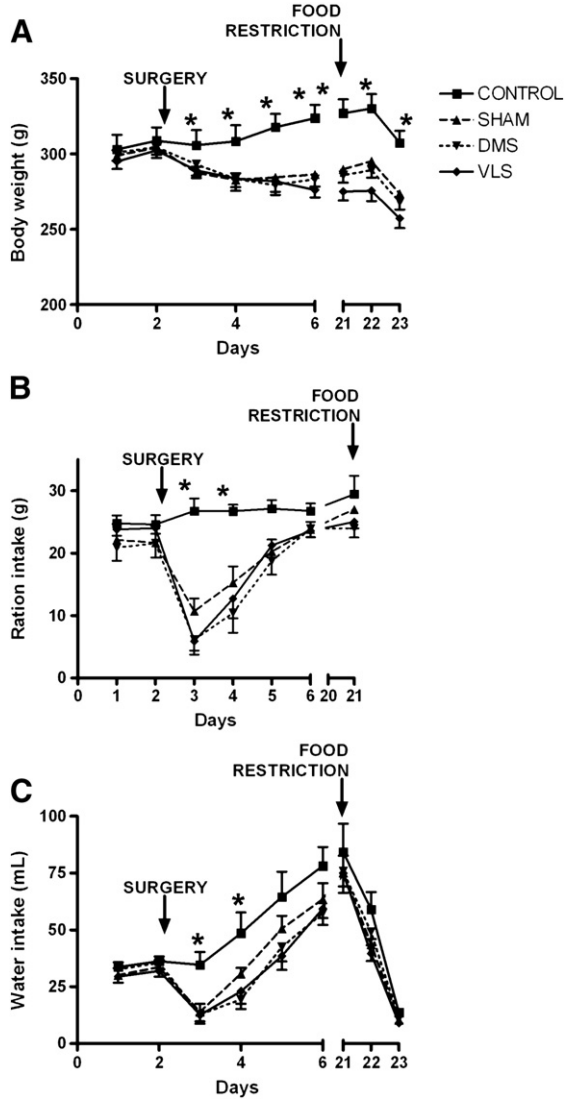

Fig. 3. The body weight, feeding and drinking behavior of the control non-operated (n = 8), ventrolateral caudoputamen (VLCP) sham-lesioned (n = 7) and NMDA-lesioned (n = 6), and dorsomedial caudoputamen (DMCP) NMDA-lesioned (n = 5) groups are expressed as mean ± SEM. * Differs significantly from the other groups, $P<0.05$.

when trying to hold the prey and failed to immediately deliver the killing bite to the head, but instead, bit other regions of the prey's body, leaving the roaches alive and moving for longer periods, and therefore, more likely to escape. In short, it is clear that the stereotyped sequence of motor actions for capturing and killing the prey had been lost in VLCP NMDA-lesioned animals. However, as noted for the other experimental groups, VLCP NMDA-lesioned animals were still able to present dodging behavior while consuming the prey, but we have not quantified these responses.

The Kruskall–Wallis test also indicated statistical differences among the experimental groups for the time spent eating the roaches [$H(3,25) = 11.85$; P,0.01], and pairwise comparisons (Dunn's multiple comparison test) revealed that VLCP NMDA-lesioned animals presented a significant drop in the time spent eating when compared to the other groups ($P<0.05$), which did not differ statistically ($P>0.05$) (Fig. 4C). Finally, animals from all experimental groups seemed equally engaged in predatory hunting during the 15-min observation period, and the Kruskal–Wallis test revealed that there was no difference among the experimental groups in the time spent in other behaviors than hunting [$H(3,25) = 2.243$; $P = 0.52$] (Fig. 4D).

As shown in Table 1, the open-field test revealed no significant alteration in locomotor activity and emotional behaviors: (total number of crossings, [$F(3,22) = 0.48$, $P = 0.69$), number of rearings $F(3,22) = 1.58$, $P = 0.22$]; time spent in grooming behavior [$F(3,22) = 2.10$, $P = 0.12$]; time of freezing [$F(3,22) = 1.48$, $P = 0.24$]; number of fecal boluses [$F(3,22) = 0.21$, $P = 0.88$]. Moreover, DMCP and VLCP NMDA-lesioned rats did not present turning behavior and the number of times they crossed the central area of the arena did not significantly differ from the control groups [$F(3,22) = 0.89$, $P = 0.46$]. Finally, it is worth commenting that animals receiving NMDA lesions in the caudoputamen did not present any abnormal behavior or seizures.

4. Discussion

The present study confirms previous evidence that the ventrolateral caudoputamen (VLCP) is involved in the selection of the proper sequence of actions performed during insect hunting.

Previous studies suggest that the VLCP occupies a strategic position in the circuit mediating predatory hunting. On the afferent side, via projections from the parafascicular thalamic nucleus, the VLCP seems to be largely influenced by the lateral part of the intermediate layer of the superior colliculus (SCig) [12]. Of particular relevance for the predatory context, neurons in the lateral SCig respond chiefly to contralateral vibrissal stimulation and small dark moving objects in the lower rostral and lateral visual field, such as the moving prey [13]. Moreover, the VLCP is targeted by dopaminergic inputs from the retrorubral field, which is heavily innervated by the medial part of the central amygdalar nucleus [14], working as an output way station from an amygdalar circuit involved in relaying information regarding the prey's odor and taste, which serve as critical motivational values to drive the predatory behavior (see [2]). Therefore, it is plausible to suggest that the prey's odor and taste may eventually induce dopamine release in the VLCP. Taken together, the evidence suggests that the VLCP is in a position to integrate vibrissal inputs and information regarding the velocity and displacement of prey, seemingly via glutamatergic inputs, as well as hedonic aspects related to prey taste and odor, via dopaminergic inputs. Other studies have also revealed that neurons in the rat VLCP are activated during oral actions, such as licking, biting, and paw-to-mouth movements [15,16].

On the motor side, it has been shown that dopaminergic or serotonergic drugs into the VLCP, but not in other regions of the caudoputamen, produce intense stereotypy, consisting of bar biting, self-biting and repetitive paw-to-mouth movements, without affecting locomotion and other kind of movements [15,17]. According to the present findings, the VLCP, and not other parts of the caudoputamen, seems to be involved in implementing the sequential pattern of action during predatory hunting. Thus, similar to non-lesioned animals, DMCP NMDA-lesioned animals presented a clear stereotyped sequence of actions and a coordinate use of the forepaws and mouth to capture and kill the prey. In sharp contrast, rats with bilateral VLCP NMDA lesions first tried to capture the prey by repeatedly pinning them against the floor with the forepaws (a pattern of action usually not seen in normal animals), and next attempted to hold the prey using the mouth with little assistance from the forepaws. They were also very clumsy to hold the prey and very often failed to deliver the killing bite to the head. The present analysis also revealed that VLCP NMDA-lesioned animals did not affect regular feeding, since, after the surgery, weight gain did not differ from the sham- and DMCP NMDA-lesioned animals. Moreover, as revealed in the open-field test, neither the VLCP nor the DMCP NMDA-lesioned animals presented any other noticeable motor impairment. The present findings are in line with the idea that the striatum is a key structure for choosing actions or sequence of actions [5–7], and thus have been pointed at as involved in sequential behavior, like learned serial reaction time [5], grooming and other kinds of stereotyped behaviors [8], such as, in

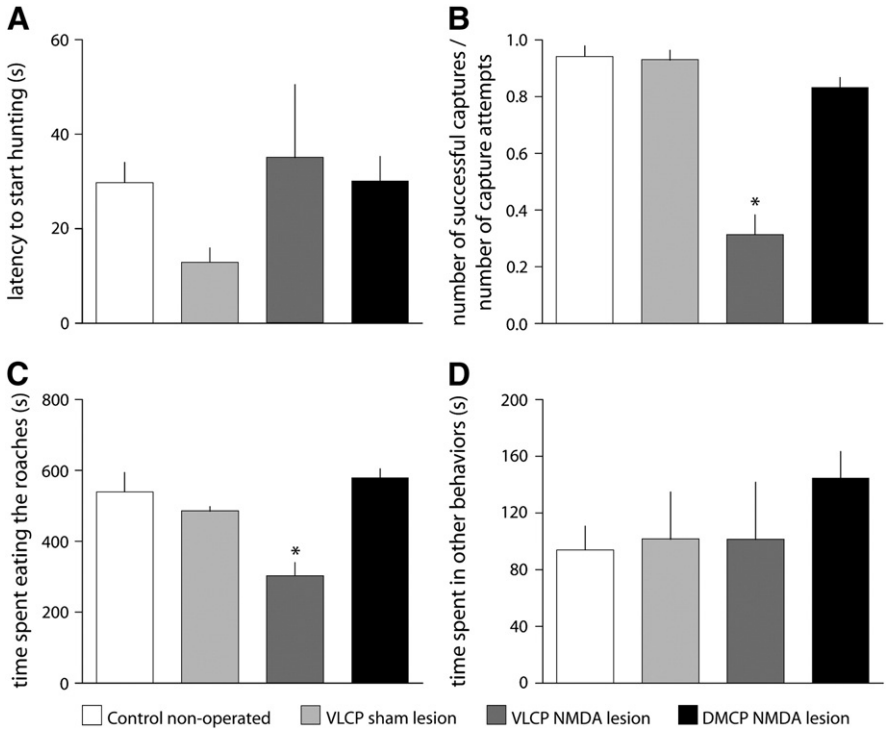

Fig. 4. Behavioral analysis. The behavioral analysis shows the latency to start hunting (A), the ratio between the number of successful captures and the number of capture attempts (B), time spent eating (C), and time spent in other behaviors (D), for the control non-operated (n = 8), ventrolateral caudoputamen (VLCP) sham-lesioned (n = 7) and NMDA-lesioned (n = 6), and dorsomedial caudoputamen (DMCP) NMDA-lesioned (n = 5) groups. Data are expressed as mean ± SEM. *Differs significantly from the other groups, $P<0.05$.

the present case, the stereotyped motor procedure during prey hunting. Although we have not checked the grooming stereotyped sequence, the amount of grooming behavior did not differ among the experimental groups.

Considering the neural circuitry related to the VLCP, the VLCP is likely to implement the motor pattern seen during prey capture through a pathway involving the lateral part of the substantia nigra pars reticulata and the lateral SCig [18]. Supporting this idea, similar to what has been found for VLCP lesions, lateral SCig lesions also disrupted the stereotyped sequence of actions seen for capturing, holding and killing the prey, in addition to influencing orienting movements toward the moving prey and the motivational drive to hunt [12]. The lateral SCig is largely known to influence the motor output through a crossed descending pathway that follows the predorsal bundle and provides extensive contacts in the pontine-medullary reticular formation and spinal cord, a pathway associated with the production of orienting pursuit-like movements [19,20]. In addition, through the connections to a number of dorsal thalamic targets (i.e., the parafascicular nucleus, the ventral medial thalamic nucleus, the ventral anterior-lateral complex of the thalamus, and the posterior complex of the thalamus), the lateral SCig may also have access to the motor and somatosensory cortex [12]. In this regard, it is noteworthy that, at least in part, these projections to the thalamic targets may arise from collaterals of the descending crossed path [21,22], which are likely to supply the efferent copy of the motor command that the superior colliculus sends to premotor sites in the brainstem and spinal cord.

Table 1
Open-field test.

	Control	Sham	DMCP	VLCP
Total number of crossings (total)	116 ± 8	133 ± 23	112 ± 21	103 ± 18
Number crossings in the central area of the arena	17 ± 2	25 ± 4	20 ± 6	16 ± 5
Number of rearings	14 ± 2	15 ± 2	20 ± 7	9 ± 3
Time spent in grooming (s)	12 ± 3	13 ± 2	27 ± 8	19 ± 7
Time of freezing (s)	11 ± 4	5 ± 3	7 ± 5	19 ± 9
Number of fecal boluses	2 ± 0.7	3 ± 0.7	3 ± 0.7	3 ± 0.9

No significant differences (ANOVA) among the control non-operated (n = 8), ventrolateral caudoputamen (VLCP) sham-lesioned (n = 7) and NMDA-lesioned (n = 6), and dorsomedial caudoputamen (DMCP) NMDA-lesioned (n = 5) groups was observed for the open-field scores, expressed as mean ± SEM.

According to our previous observations, reversible blockade with lidocaine in the VLCP increased the latency to start hunting, reflecting a possible influence in the motivational drive to attack the roaches. This finding could not be confirmed in the present study, where VLCP NMDA-lesioned animals started chasing the prey shortly after they had been delivered into the testing box, and, compared to the other experimental groups, presented no differences in the latency to start hunting. In addition, during the observation period, the time spent in behaviors other than hunting (another parameter that could reflect a possible loss of interest in chasing the prey) did not differ among VLCP NMDA-lesioned animals and other experimental groups. The reason for this discrepancy between reversible blockade with lidocaine inactivation and NMDA lesions may possibly be related to compensatory neuronal effects likely to arise during post-lesion recovery and prevent an effect on the circuits mediating the motivational drive to hunt. Previous studies suggest that the lateral part of the periaqueductal gray seems to be a nodal part of a neural circuit involved in the decision-making process between hunting/ foraging and other behavioral responses [23,24]. It has been previously shown that NMDA cytotoxic lesions of the lateral PAG, but not other parts of the PAG, produced a dramatic effect in inhibiting insect hunting, an effect thought to be mediated through the lateral PAG projections to the lateral hypothalamic region containing orexin neurons [23,24]. Among the elements of the neural network controlling predatory behavior, the lateral SCig (involved in detecting prey displacements) and the central nucleus of the amygdala (the main output way station for the amygdalar circuit involved in detecting prey's odor and taste) provide extensive projections to the lateral part of

the periaqueductal gray, and are thus likely to influence the motivational drive to hunt [24,25]. In line with this view, bilateral NMDA lesions of the lateral SCig have also been shown to drastically increase the latency to start hunting [12].

Overall, the present study emphasizes the role of the VLCP as a locus to implement the stereotyped sequence of actions seen during prey capture and handling, but the study did not confirm any VLCP role in influencing the motivational drive to hunt. Together with other studies from our group, the present work serves as an important piece of information that helps to reveal the neural systems underlying predatory hunting.

Acknowledgments

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